HUMAN HEALTH EFFECTS OF WILDLAND SMOKE

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Introduction

Smoke from wildfires and prescribed fires contain many hazardous chemicals and pose a significant public health threat. Fine particulate matter, PM2.5 (<2.5 micrometers) is most associated with causing adverse effects. Adverse health impacts can occur from both short-term smoke exposures (lasting hours to days) and long-term exposures.

"Even though woodsmoke [including wildland smoke] *is natural, it is not benign. Indeed, there is a considerable and growing body of epidemiologic and toxicologic evidence that both acute and chronic exposures to woodsmoke in developed country populations, as well as in the developing world, are associated with adverse health impacts. Woodsmoke contains thousands of chemicals, many of which have well-documented adverse human health effects, including such commonly regulated pollutants as fine particles, CO* [carbon monoxide], *and nitrogen oxides as well as ciliatoxic respiratory irritants such as phenols, cresols, acrolein, and acetaldehyde; carcinogenic organic compounds such as benzene, formaldehyde, and 1,3 butadiene; and carcinogenic cyclic compounds such as PAHs* [polyaromatic hydrocarbons]. *Woodsmoke contains at least five chemical groups classified as known human carcinogens by the International Agency for Research on Cancer (IARC), others categorized by IARC as probable or possible human carcinogens, and at least 26 chemicals listed by the U.S. EPA as hazardous air pollutants. Among the currently regulated pollutants in woodsmoke, fine particles (PM2.5) serve as the best exposure metric in most circumstances and, in addition, tend to be among the most elevated in relation to existing air quality standards"* (Naeher2007).

Wildfire and prescribed fire smoke also contain heavy metals, including mercury, as well as radionuclides. According to Carvalho, et al., forest fire smoke contains radionuclides at levels that can be greater than those in cigarette smoke (Carvalho2014).

Recent research has also found viable bacteria and fungi in wildland fire smoke (Kobziar2018), at levels above those present before burning occurred (Mirskaya2020). It has been hypothesized that these microorganisms could represent an infectious risk to the public. In 2019, researchers linked California wildfires with increased hospitalizations for invasive mold infections, including Aspergillus mold and Coccidioides fungus (causes Valley Fever) (Mulliken2019).

There is evidence that wildland smoke is more toxic than typical urban air pollution (Jaffe2020). Wildfire particulate matter tends to have a smaller particle size and contain more oxidative and proinflammatory components than urban particulates (Xu2020).

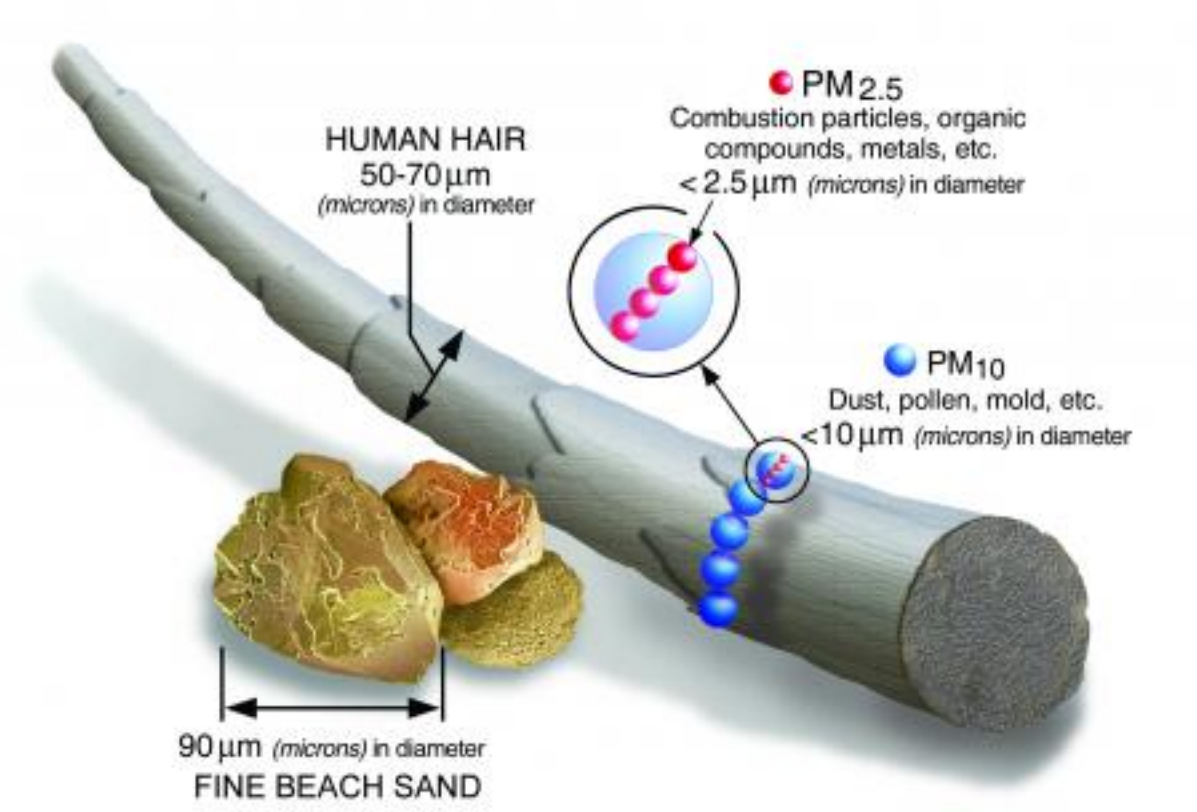
Exposure to wildfire and prescribed fire smoke can cause irritation of the eyes, nose, throat; wheezing, coughing, and shortness of breath; and headache. It can also aggravate lung disease, like asthma and chronic obstructive pulmonary disease

(COPD), and cardiovascular conditions. It is not unusual for people with chemical sensitivities to also experience severe fatigue, increased body pain, and brain "fog."

Wildland fire smoke can also have long-lasting effects on human health. Orr, et al., found a significant decrease in lung function among many community members one year following a wildfire event, and this remained decreased two years following the smoke exposure (Orr2020). Landguth, et al. found that higher daily average PM2.5 concentrations during a wildfire season was positively associated with increased influenza in the following winter influenza season (Landguth2020).

Particulate Matter (PM)

What is PM, and how does it get into the air?



Size comparisons for PM particles

PM stands for particulate matter (also called particle pollution): the term for a mixture of solid particles and liquid droplets found in the air. Some particles, such as dust, dirt, soot, or smoke, are large or dark enough to be seen with the naked eye. Others are so small they can only be detected using an electron microscope.

Particle pollution includes:

- **PM10:** inhalable particles, with diameters that are generally 10 micrometers [microns]

and smaller; and

- **PM2.5**: fine inhalable particles, with diameters that are generally 2.5 micrometers [microns] and smaller.

Sources of PM

These particles come in many sizes and shapes and can be made up of hundreds of different chemicals.

Some are emitted directly from a source, such as construction sites, unpaved roads, fields, smokestacks or fires.

Most particles form in the atmosphere as a result of complex reactions of chemicals such as sulfur dioxide and nitrogen oxides, which are pollutants emitted from power plants, industries and automobiles.

[www.epa.gov/pm-pollution/particulate-matter-pm-basics]

What are the Harmful Effects of PM?

Particulate matter contains microscopic solids or liquid droplets that are so small that they can be inhaled and cause serious health problems. Some particles less than 10 micrometers in diameter can get deep into your lungs and some may even get into your bloodstream. Of these, particles less than 2.5 micrometers in diameter, also known as fine particles or $PM_{2.5}$, pose the greatest risk to health.

The size of particles is directly linked to their potential for causing health problems. Small particles less than 10 micrometers in diameter pose the greatest problems, because they can get deep into your lungs, and some may even get into your bloodstream.

Exposure to such particles can affect both your lungs and your heart. Numerous scientific studies have linked particle pollution exposure to a variety of problems, including:

- premature death in people with heart or lung disease
- nonfatal heart attacks
- irregular heartbeat
- aggravated asthma
- decreased lung function
- increased respiratory symptoms, such as irritation of the airways, coughing or difficulty breathing.

People with heart or lung diseases, children, and older adults are the most likely to be affected by particle pollution exposure.

[www.epa.gov/pm-pollution/health-and-environmental-effects-particulate-matter-pm]

Both coarse particles (< 10 micrometers) and fine particles (<2.5 micrometers) enter the lungs and induce an inflammatory response. Fine particles can be absorbed into the blood stream and cause inflammation in all parts of the body.

One study found that PM samples collected during a wildfire event were more toxic than the same amount of PM from normal ambient air (Wegesser2009).

Another study notes "*there is some evidence to suggest that PM2.5 from wildfires may have a stronger adverse effect on respiratory morbidity at the same levels [emitted by other sources], and that there is a difference in toxicological response based on particulate matter source*" (Alman2016).

Shi, et al. found that both short- and long-term exposures to PM2.5 in a Medicare population were associated with all-cause mortality, even for exposure levels not exceeding U.S. EPA standards (12microgram/m3 annual average, 35 microgram/m3 daily). In addition, the association between short-term exposure and mortality appeared to be linear across the entire exposure distribution, indicating there was no safe level of exposure. (Shi2016).

Elliott, et al., compared dispensations for salbutamol [used to treat asthma] in forest fire-affected and non-fire-affected populations in British Columbia, Canada. Fire season PM2.5 was positively associated with salbutamol dispensations in all fire-affected populations (Elliott2013).

Sensitive Populations

Inhaling smoke is not good for anyone, even healthy people, but there are many populations at increased risk of harm from air pollution.

The following data on groups at risk from exposure to air pollution are provided by the American Lung Association in New Mexico (www.lung.org/our-initiatives/healthy-air/sota/city-rankings/states/new-mexico).

The total population of Santa Fe County is 150,358.

Under 18 years of age	26,394 (18%)
65 years and over	38,106 (25%)
Pediatric asthma	1,437 (1%)
Adult asthma	10,524 (7%)
COPD (chronic obstructive pulmonary disease)	7,951 (5%)
Lung cancer	51 (negligible)
Cardiovascular disease	11,248 (8%)
Poverty estimate	18,378 (12%)

It is also known that air pollution affects pregnant women. On average, pregnant women account for approximately 1% of a total population.

Those with chemical sensitivities are also at greater risk of harm from air pollution and can have serious physical reactions to exposures to even minute amounts of pollutants. A 1997 survey conducted by the NM Department of Health found that 16% of the state's respondents reported being unusually sensitive to everyday chemicals and 2% reported they had been diagnosed with multiple chemical sensitivities (MCS). The most recent national prevalence study found 25.9% of respondents reported being chemically sensitive and 12.8% of reported having been medically diagnosed with MCS (Steinemann2018).

Taken all together the above percentages of vulnerable populations totals 103%. Even though there are overlaps in these categories, it is clear that a significant portion of the population, possibly even the majority of the population, is at increased risk of harm from exposure to wildland fire smoke.

Respiratory Effects

Why is particle pollution a respiratory health concern?

Studies have linked particle pollution exposure to a variety of respiratory health effects, including:

- Respiratory symptoms including cough, phlegm, and wheeze.
- Acute, reversible decrement in pulmonary function.
- Inflammation of the airways and lung (this is acute and neutrophilic).
- Bronchial hyperreactivity.
- Acute phase reaction.
- Respiratory infections.
- Respiratory emergency department visits.
- Respiratory hospitalizations.
- Decreased lung function growth in children.
- Chronic loss of pulmonary function in adults.
- Asthma development.
- Premature mortality in people with chronic lung disease.

People with heart or lung disease, older adults, children, people with diabetes, and people of lower SES [socioeconomic status] are at greater risk of particle pollution-related health effects.

Though the respiratory system has remarkable resilience to air pollution via its repeated mobilization of defense and repair mechanisms, constant exposure to

elevated particle pollution will contribute to reduced respiratory function, even in apparently healthy people. Therefore, although we cannot completely avoid particle pollution exposure, taking simple steps to reduce exposure will reduce the severity of lung and systemic adverse health effects in both healthy and more sensitive people.

How does particle pollution affect the respiratory system?

Particles deposited in the respiratory tract in sufficient amounts can induce inflammation, which has been demonstrated in both animal and controlled human exposure studies. The extent of pulmonary inflammation depends on particle dose and composition. Controlled human exposure studies have demonstrated increased markers for pulmonary inflammation following exposure to a variety of different particle types. For example, organic carbon particles and transition metals from combustion sources can elicit a strong inflammatory response (U.S. EPA, 2009).

Airway inflammation increases airway responsiveness to irritants (e.g., cold air, particle pollution, allergens, lipopolysaccharides, and gaseous pollutants) and may reduce lung function by causing bronchoconstriction. At a cellular level, inflammation may damage or kill cells and compromise the integrity of the alveolar-capillary barrier. Repeated exposure to particle pollution aggravates the initial injury and promotes chronic inflammation with cellular proliferation and extracellular matrix reorganization (Berend, 2016).

Mobilization of the pulmonary immune system and other defense mechanisms is essential in the response to particle pollution. The overall balance between injury (inflammatory activity) and repair (anti-inflammatory defenses) plays an important role in the pathogenesis and progression of inflammatory respiratory diseases such as asthma. Inhalation of particle pollution may affect the stability or progression of these conditions through inflammatory effects in the respiratory tree.

What are the respiratory effects of acute exposure?

Studies have reported respiratory effects related to acute exposure to fine particles, including respiratory symptoms (especially in children and those diagnosed with asthma), reduction in pulmonary function, and increased airway inflammation and responsiveness. Additionally, epidemiologic studies have demonstrated that respiratory effects associated with particle pollution can be serious enough to result in emergency department visits and hospital admissions, including COPD and respiratory infections.

The relationship between exposure to ambient particle pollution concentrations and adverse respiratory effects was clearly demonstrated in a series of studies conducted in the Utah Valley by Pope (1989, 1991). When a steel mill, which was the source of 90 percent of local particle pollution emissions in the Utah Valley, was out of operation for one year, hospital admissions for bronchitis and asthma in the valley decreased by almost 50 percent and were comparable to those in other regions not polluted by the mill. Once mill operation resumed, hospital admissions increased. The mortality rate in the valley showed a similarly positive association with particle pollution levels during the same period.

The combination of experimental and epidemiologic studies has provided evidence of a relationship between short-term (daily) exposures to particle pollution and a number of respiratory-related effects, including elevated morbidity, higher frequency of emergency department visits and hospital admissions, as well as excess mortality. Often people with pre-existing diseases are at greatest risk for potential respiratory-related health effects due to short-term particle exposures (Ling and van Eeden, 2009).

What are the respiratory effects of chronic exposure?

Epidemiologic studies conducted in the U.S. and abroad provide evidence of associations between long-term exposure to fine particles and both decrements in lung function growth in children and increased respiratory symptoms.

The Children's Health Study (Gauderman et al., 2015) evaluated three separate cohorts of children who had longitudinal lung-function measurements recorded over the same 4-year age range (11 to 15 years) and in the same five study communities but during different calendar periods. The study shows an association between improvements in air quality in southern California and measurable improvements in lung-function development in children. Improved lung function (mean attained FEV1 and FVC values at 15 years of age) was most strongly associated with lower levels of particle pollution (PM2.5 and PM10) and nitrogen dioxide. These associations were observed in boys and girls, Hispanic white and non-Hispanic white children, and children with asthma and children without asthma, which suggests that all children have the potential to benefit from reduced exposure to particle pollution.

This same group conducted another epidemiological study that looked at the impact of improvement in particle pollution levels in Southern California between 1993 and 2012. It found that as ambient pollution levels improved there was a statistically significant decrease in bronchitis symptoms in children, especially among those with asthma (Berhane et al. 2016).

How does particle pollution affect people with asthma?

According to 2014 data, approximately 24 million Americans have asthma-- about 1 in 12 children (8.6 percent) and 1 in 14 adults (7.4 percent)(CDC, 2016). (For the most recent asthma data, go to CDC's asthma data page.) Asthma is a disease characterized by a variable degree of chronic airway inflammation associated with airway hyper-reactivity, reversible bronchoconstriction (used as an index of severity), and excessive mucus production. These abnormalities lead to symptoms and signs of asthma that include episodes of wheezing, coughing, chest tightness, and dyspnea. Asthma symptoms can be triggered by numerous environmental factors that can lead to bronchoconstriction and aggravate the disease. These environmental factors include exercise, humidity, temperature, allergens, viral infection, stress, and inhalation of air pollutants. Sensitivity to specific environmental triggers varies between individuals.

Several factors may cause people with asthma to be at increased risk of particle pollution-related health effects compared to healthy individuals.

- Airway hyper-reactivity and bronchoconstriction can affect particle deposition in a number of ways. Deposition can be increased in the conducting airways and some peripheral regions as a result of both obstruction and increased air flow to the better ventilated areas of the lung.
- Most particle pollution is pro-inflammatory and can aggravate pre-existing airway inflammation, which increases pro-inflammatory mechanisms and accelerates the inflammatory cascade.
- Allergens are a major factor in asthma development and exacerbation. The intensity of asthma symptoms and bronchial responsiveness varies with allergen sensitization, and people with allergic asthma are at increased risk for particle pollution-related health effects during times of high-allergen exposure (Silverman et al., 1992).

Biological particles (i.e., microbes, viruses, and spores) may lead to asthma exacerbation by aggravating inflammation and causing infection. In general, epidemiologic data provide substantial evidence for the association between particle pollution exposure and adverse effects in individuals with allergies and asthma, as assessed by frequency and severity of respiratory symptoms, pulmonary function changes, medication use, and ambient particle pollution levels. There is evidence that both the development of asthma and its exacerbation can be associated with particle pollution exposure.

What are the health disparities for asthma?

Asthma effects are more problematic in young children, older adults, minorities, and those with lower SES [socioeconomic status]. Minority children have higher

prevalence of asthma and higher rates of asthma-related emergency room visits, hospitalizations, and deaths than white children. Environmental factors related to SES may be important in contributing to these asthma disparities. For example, poor inner city children with asthma may be at increased risk from air pollution because they live near high-density traffic or industrial sources of particle pollution or because they have poor indoor air quality due to housing conditions. Because such children may have limited access to medical services and asthma education, these effects may be magnified (Gold et. al, 2005).

Children with asthma seem to be more affected by particle pollution than adults with asthma. This may be in part due to anatomical factors that lead to higher deposition of particle pollution in the tracheobronchial region of the lung in children. Other proposed factors that contribute to children being at increased risk of particle pollution-related health effects include behavioral factors such as increased exercise and time outdoors.

How does particle pollution affect people with COPD?

COPD is a major cause of disability and is the third leading cause of death in the United States (Ford et al, 2013). COPD is a lung disease characterized primarily by chronic airway inflammation, mucous hypersecretion, and progressive airflow limitation. These structural changes result in symptoms of cough, dyspnea, and increased sputum production. COPD comprises a spectrum of clinical disorders that include emphysema, bronchiectasis, and chronic bronchitis. COPD risk factors are both genetic and environmental. Elevated particle pollution contributes to the exacerbation of this disease and likely its pathogenesis. The role of other factors, such as developmental factors, is not well understood.

Like people with asthma, people with COPD are at greater health risk from particle pollution exposure than healthy individuals. There is a substantial overlap between the asthma and COPD phenotypes. The key underlying mechanisms are:

- Airway inflammation dominated by neutrophilic infiltration of the airways is aggravated by pro-inflammatory particle pollution.
- Increased sputum production combined with variable airway narrowing and uneven ventilation produces heterogeneous particle deposition, which creates localized regions (hot spots) with excessive particle accumulation. This accumulation, when combined with reduced particle clearance, substantially increases the probability of tissue injury beyond inflammation (Kim and Kang, 1997).

A few controlled human exposure studies of elderly COPD patients reported an

association between respiratory effects and fine particle pollution. Even fewer studies have explored the effects that ambient particle pollution may have on COPD development.

Epidemiological panel studies exploring the potential relationship between daily particle pollution levels and respiratory effects in people with COPD reported increased symptomatic response, increased use of evening medication (winter time), and small decrements in spirometric lung function in the days immediately following elevated particle pollution (PM10 and PM2.5) levels. Other endpoints showed an inconsistent response (Silkoff et al., 2005, Pope and Kanner, 1993). Though the induced effects may be insignificant, frequent exacerbation of symptoms and lung function impairment may accelerate COPD progression.

Time-series studies appear to show evidence of an association between acute exposures (i.e., daily) to particle pollution and morbidity (i.e., emergency department visits and hospital admissions) and mortality among individuals with COPD.

What is the role of fine particles in lung cancer incidence and mortality?

Prior to discussing the relationship between particle exposure and lung cancer, it is important to note the evolving scientific evidence. In the context of EPA, the evaluation of scientific evidence for cancer and other health effects for particle pollution occurs in an Integrated Science Assessment (ISA) as part of the National Ambient Air Quality Standards (NAAQS) review process.

The 2009 ISA (the most recent ISA for particle pollution) describes that epidemiologic studies generally demonstrated consistent positive associations between fine particle exposure and lung cancer mortality, but studies generally did not report associations between fine particles and lung cancer incidence (Pope et al., 1995; Dockery et al., 1993). Evidence from toxicological studies indicated that various combustion-related sources (e.g., wood smoke, coal combustion) are mutagenic and genotoxic, which provides biological plausibility for the effects observed in epidemiologic studies, and some components of particle pollution are known human carcinogens (e.g., specific arsenic, cadmium and chromium compounds).

More recently, the International Agency for Research on Cancer (IARC) conducted an evaluation on the carcinogenicity of outdoor air pollution, including particle pollution, and concluded that both are Group I agents (carcinogenic to humans). This IARC review focused on all routes of exposure and included an evaluation of individual components of particle pollution that are known human carcinogens.

Since 2009, there has been a dramatic increase in the number of epidemiologic studies that have examined chronic particle pollution exposures and both lung cancer incidence and mortality. Many of these studies are summarized in a meta-analysis by Hamra et al. (2014) that provide evidence of a relationship between fine particle exposure and lung cancer incidence and mortality. As part of the upcoming review of decisions to retain or revise the NAAQS for particle pollution, the EPA recently began an evaluation of evidence for cancer and other health effects resulting from particle pollution exposures that has been published since completion of the 2009 ISA. Information pertaining to publicly available drafts of EPA evaluations of the scientific evidence for particle pollution and lung cancer and other health effects can be found at EPA's Integrated Science Assessments website.

[www.epa.gov/particle-pollution-and-your-patients-health/health-effects-pm-patients-lung-disease]

Finlay et al. states that a review of the published evidence shows that human health can be severely affected by wildfires and that wood smoke has high levels of particulate matter and toxins. According to the authors, respiratory morbidity predominates, but cardiovascular, ophthalmic and psychiatric problems can also result (Finlay2012).

A study by Henderson, et al., found that forest fire smoke was associated with increases in self-reported symptoms, medication use, outpatient physician visits, emergency room visits, hospital admissions, and mortality. The associations were strongest for the outcomes most specific to asthma (Henderson2012).

U.S. Environmental Protection Agency (EPA) researchers investigated the relationship of PM2.5 levels with emergency department visits and acute hospitalizations for respiratory and cardiovascular outcomes during the 2012 Colorado wildfires. They found a positive association between PM2.5 and respiratory diseases, supporting evidence from previous research that wildfire PM2.5 is an important source for adverse respiratory health outcomes. (Alman2016).

Hutchinson, et al., examined the healthcare utilization of Medi-Cal recipients during the fall 2007 San Diego wildfires. They found that respiratory diagnoses, especially asthma, were elevated during the wildfires; wildfire-related healthcare utilization appeared to persist beyond the initial high-exposure period; increased adverse health events were apparent even at mildly degraded Air Quality Index levels; young children had bigger increases in healthcare visits during the peak fire period than older age groups; and very young children aged 0-1 were the most impacted experiencing a 243% increase in healthcare visits (Hutchinson2018).

Recently, Stowell, et al., studied the associations of wildfire smoke PM2.5 exposure with cardiorespiratory events in Colorado from 2011-2014. The authors found that for every 1 microgram/m3 increase in fire smoke PM2.5, statistically significant associations were observed for asthma and combined respiratory disease. Yet despite these associations, there was an absence of association with *total* PM2.5 concentrations. The authors state their findings point to potential toxic differences between smoke and non-smoke PM2.5 exposure, suggesting that PM2.5 from wildfire smoke could pose a significant threat to public health (Stowell2019).

Liu, et al., investigated wildfire-specific fine particulate matter and the risk of hospital admission in urban and rural counties. They found an increase in risk of respiratory admission during smoke wave days with high wildfire-specific PM2.5 (>37 micrograms/m3) compared to matched non-smoke wave days. They also concluded that "*Respiratory effects of wildfire-specific PM2.5 may be stronger than that of PM2.5 from other sources"* (Liu(a)2017).

Black, et al., evaluated the current literature on wildfire smoke and human health. The authors state that wildfire smoke has a distinct composition compared to other sources of air pollution. Wildfires produce proportionately more fine (under 2.5 microns) and ultrafine (under 1 micron) particulate, compared to coarse particulate, defined as particles fewer than 10 microns in size (PM10). The authors also note that wildfires also have a long smoldering phase, as wildfire containment strategies focus on extinguishing the flame phase while the smoldering phase is left to burn itself out, sometimes for months after a fire is considered contained. The smoldering phase of wood burning is associated with higher output of particulates, and can account for a large proportion of the total wildfire air pollutant emissions (Black2017).

Delfino, et al., studied the relationship of respiratory and cardiovascular hospital admissions during southern California wildfires of 2003. They found wildfire-related PM2.5 led to increased respiratory hospital admission, especially for asthma (Delfino2009).

Cascio addressed wildland fire smoke and human health. He states that systematic reviews conclude that a positive association exists between exposure to wildfire smoke or wildfire particulate (PM2.5) and all-cause mortality and respiratory morbidity. Respiratory morbidity includes asthma, chronic obstructive pulmonary disease (COPD), bronchitis and pneumonia. Susceptible populations include people with respiratory and possibly cardiovascular diseases, middle-aged and older adults, children, pregnant women and the fetus. The size of the population at risk from wildland fire smoke is increasing. Wildland fire smoke represents a costly and growing global public health problem. Studies have shown evidence that risks are greater for older women, African-Americans, and those with indicators of lower socio-economic status (Cascio2018).

Reid, et al., investigated health effects associated with fine particulate matter during 2008 wildfires in northern California. They observed a linear increase in risk for asthma hospitalizations and asthma emergency department (ED) visits with increasing PM2.5 during the wildfires. ED visits for chronic obstructive pulmonary disease (COPD) were associated with PM2.5 during the fires and this effect was significantly different from that found before the fires (Reid2016).

Roscioli, et al., employed models of human airway epithelium exposed to wildfire smoke-extract to examine changes in airway epithelial cell survival, fragility and barrier function. Primary epithelial models exposed to wildfire smoke-extract exhibited a significant blockade in autophagy, significant PARP cleavage indicative of apoptotic changes, and barrier dysfunction with significant increases in paracellular molecular permeability and reduction of tight junction proteins. These cultures also exhibited increased IL-6 secretion consistent with the aberrant and pro-inflammatory repair response observed in chronic obstructive pulmonary disease (COPD) airways. Further, blocks in autophagy and barrier disruption were significantly elevated in response to wildfire smoke-extract in comparison to similar exposure with cigarette smoke-extract (Roscioli2018).

Cardiovascular Effects

Why is particle pollution a cardiovascular health concern?

Cardiovascular disease accounts for the greatest number of deaths in the United States. One in three Americans has heart or blood vessel disease. According to the American Heart Association (AHA), one in every three deaths is attributed to cardiovascular disease, and expenses related to cardiovascular disease represent 17 percent of overall national health expenditures (Heidenreich et al., 2011).

Traditional risk factors for cardiovascular disease, such as male gender, age, increased blood pressure, high cholesterol, and smoking account for about 50 percent of cardiac events. Other factors acting independently, or together with established risk factors, likely contribute to the development of cardiovascular disease. Air pollution exposure is one such risk factor and is known to exacerbate existing, and contribute to the development of, cardiovascular disease.

Evidence linking ambient particle pollution exposure and adverse effects on cardiovascular disease is particularly strong (Newby et al., 2014). The AHA concluded both that exposure to increased concentrations of fine particle pollution over a few hours to weeks can trigger cardiovascular disease-related mortality and nonfatal events and that exposures of a few years or more to increased concentrations of fine particle pollution increases the risk of cardiovascular mortality and decreases life expectancy (Brook et al., 2010).

On an individual level, the risk of cardiovascular disease from particle pollution is smaller than the risk from many other well-established factors. At the population level, acute and chronic exposure to particle pollution can increase the numbers of cardiovascular events, including hospitalizations for serious cardiovascular events, such as coronary syndrome, arrhythmia, heart failure, and stroke, particularly in people with established heart disease.

Your patients with cardiovascular disease, including those who have angina, heart failure, particular arrhythmias, or that have risk factors for heart disease (e.g., those who are smokers, obese, or older adults) may be at greater risk of having an adverse cardiovascular event from exposure to fine particles. Unlike some risk factors that contribute to cardiovascular morbidity and mortality, people can take steps to reduce their exposure to particle pollution. Ninety-two percent of patients with cardiovascular disease are not informed of health risks related to air pollution (Nowka et al., 2011). Reducing population exposure to fine particle pollution has been shown to be associated with decreases in cardiovascular mortality (even within a few years of reduced exposure) (Pope et al., 2009; Correia et al., 2013).

How does particle pollution affect the cardiovascular system?

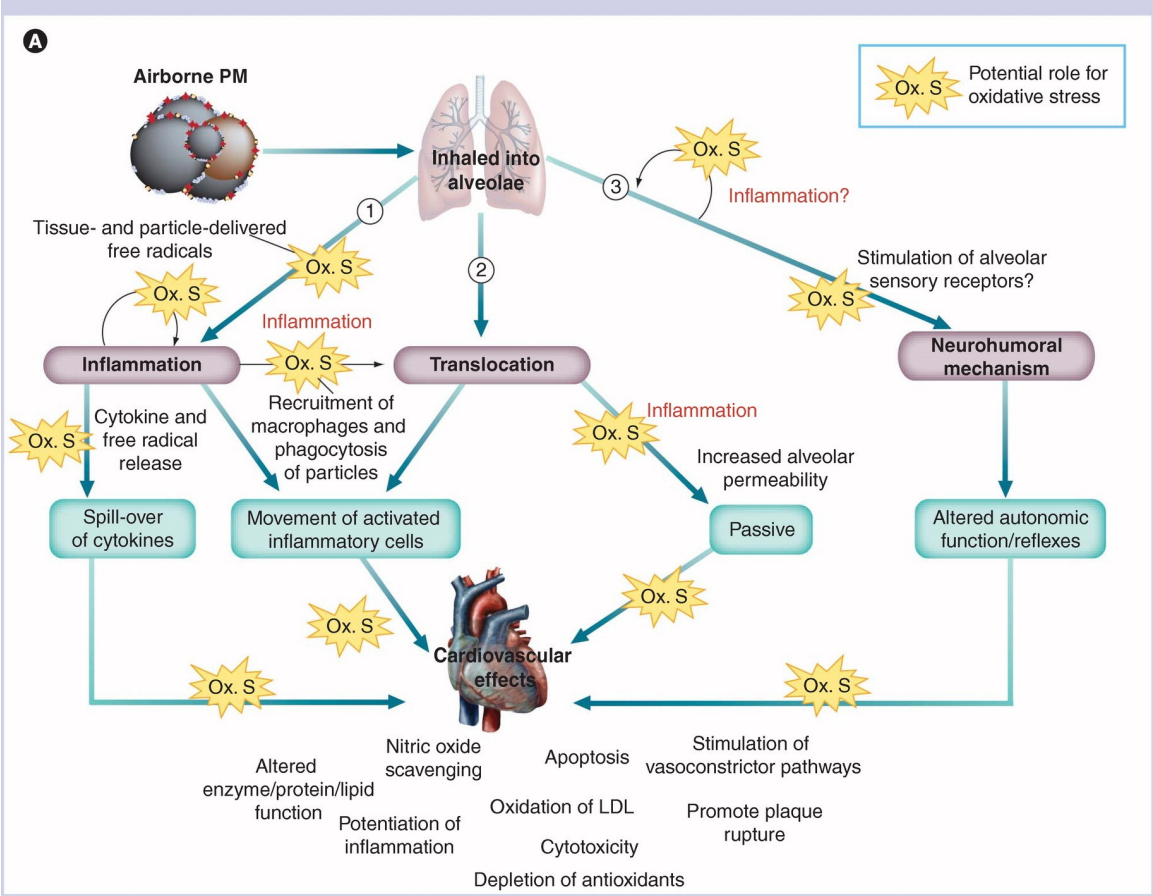
The mechanisms by which exposure to fine particle pollution can affect the cardiovascular system are under continuous examination. Exposure to inhaled fine particles appears to affect cardiovascular health through three primary pathways:

- Systemic inflammation.
- Translocation into the blood.
- Direct and indirect effects on the autonomic nervous system.

Oxidative stress is an underlying effect due to particle exposure that has been shown to impact endothelial function, pro-thrombotic processes, cardiac electrophysiology, and lipid metabolism.

The pathways by which inhaled particle pollution affects cardiovascular health are detailed in Figure 6. Inhaled particle pollution reaches the alveoli, at which point it can increase the formation of reactive oxygen species (ROS) and initiate an inflammatory response. Alveolar macrophages are likely to release pro-inflammatory cytokines with secondary effects on vascular control, heart rate variability, contractility, and rhythm. Alternatively, following deposition, small amounts (<1%) of ultrafine insoluble particles, or more soluble components of any size particles (e.g., metals), may translocate from the lung directly into the circulation where the particle might have direct impact on cardiovascular function

and/or have direct effects on the central nervous system with secondary effects on the heart and blood vessels via the autonomic nervous system.



6. Three possible mechanisms accounting for cardiovascular effects associated with particle pollution exposure. (1) Particles induce an inflammatory response in the lungs, leading to release of cytokines and other mediators that 'spill-over' into the systemic circulation. (2) Some ultrafine particles can translocate from the alveolus into the circulation and then interact directly with the heart and vasculature with or without the participation of inflammatory cells. (3) Particles might activate pulmonary sensory receptors and modulate the autonomic nervous system. Oxidative stress could play a role in exacerbating the stages of each pathway, as well as promoting interactions between pathways (e.g., in conjunction with inflammation). Reprinted with permission from Future Medicine Ltd. (Miller MR, Shaw CA, Langrish JP. 2012. From particles to patients: oxidative stress and the cardiovascular effects of air pollution. Future Cardiology 8(4):577-602).

Several studies identify an increase in inflammatory mediators and endothelial activation biomarkers after ambient particle pollution and urban air pollution

exposure (i.e., C-reactive protein (CRP), TNF-alpha, prostaglandin E2, CRP, interleukin-1b, and endothelin-1) (Pope et al., 2004; Calderón-Garcidueñas et al., 2008). Traffic-related particle pollution, which consists of a mixture of pollutants, has been shown to be positively associated with a number of subclinical effects including inflammation, oxidative stress, and autonomic nervous system balance, providing evidence that traffic-related air pollution is an important source of particle pollution (Chuang et al., 2007).

Studies using concentrated air particles provide important insights into the effects of exposure to particle pollution on cardiovascular endpoints in healthy adults. Ghio and colleagues studied the effects of either filtered air or particles concentrated from the immediate environment (averaging 120 μg/m3). After two hours of exposure, subjects underwent bronchoscopy and assessment of evidence of systemic inflammation. Exposure to fine particles produced no cardiopulmonary symptoms, yet bronchoalveolar lavage showed a mild increase in neutrophils in both the bronchial and alveolar fractions, and fibrinogen was increased the next day (Ghio et al., 2000).

What are the cardiovascular effects?

Acute and chronic exposure to fine particle pollution has been shown to increase the risk of hospitalizations for cardiovascular conditions and mortality. However, multi-city epidemiologic studies of mortality and hospital admissions have provided evidence of regional heterogeneity in risk estimates (Dominici et al., 2006; Zanobetti and Schwartz, 2009). It has often been hypothesized that the regional heterogeneity observed in epidemiologic studies may be a reflection of a number of factors including different sources and the chemical composition of fine particles varying between cities and regions, as well as demographic or exposure differences. To date, the underlying factors that contribute to this heterogeneity have yet to be identified.

Clinically important cardiovascular effects of inhaled particles include:

- Acute coronary syndrome, including myocardial infarction, unstable angina.
- Arrhythmia.
- Exacerbation of chronic heart failure.
- Stroke.
- Sudden cardiac death.

Such effects can be measured after acute exposure, and there is accumulating evidence that chronic exposure accelerates atherosclerosis and reduces life expectancy.

What are the acute exposure effects?

Population-based studies, small repeated-measure panel studies, and acute exposure studies in humans support the conclusion that inhalation of particle pollution induces small changes in blood pressure, oxygen saturation, endothelial function, systemic changes in acute phase reactants, coagulation factors, inflammatory mediators, and measures of oxidative stress. Systemic blood pressure and endothelial function changes, acute coronary syndrome (including myocardial infarction and unstable angina), increased ventricular arrhythmias in people with implantable (or internal) cardiac defibrillators (ICDs), exacerbation of heart failure, ischemic stroke, and cardiovascular mortality are all well-established clinical cardiovascular health effects associated with acute exposure to fine particles.

Blood pressure and endothelial function: Acute fine particle exposure causes a small increase in systolic and diastolic blood pressure (Liang et al. 2014). Some studies of persons without cardiovascular disease indicate a small increase in blood pressure associated with acute exposures to particle pollution (Auchincloss et al., 2008; Gong et al., 2003). Increased sympathetic tone and changes in vasomotor regulation caused by inflammation and oxidative stress are the most likely physiological changes to explain an increase in blood pressure (Brook et al., 2002). Because particle pollution is ubiquitous in the ambient air, exposures resulting in increases in blood pressure at the population level can have important public health implications (Brook, 2005).
Several studies indicate that filtering particles from the air either prevents or decreases particle-induced changes in physiological and biochemical determinants of heart and vascular health (Bräuner et al., 2008; Langrish et al., 2012). However, the clinical benefit of particle filters is not yet established.

Acute coronary syndrome: Several studies indicate that the onset of unstable angina and myocardial infarction are associated with exposure to ambient fine particle pollution (Pekkanen et al. 2002; Peters et al., 2001). Clinical studies show that particle pollution exposure increases the magnitude of ST-segment changes during ischemia, suggesting that exposure to particle pollution increases the severity of ischemia (Pekkanen et al., 2002).

Arrhythmias: An increase in ventricular and supraventricular arrhythmias in persons with ICDs (indicated by an increase in the discharge of the ICD) has been positively associated with increases in fine particle concentrations, which is supported by evidence of a linear exposure response (Peters et al., 2000; Rich et al., 2005; Dockery et al., 2005; Link et al., 2013). Stronger associations were found between air pollution and ventricular arrhythmias for episodes within a few days of a previous arrhythmia, suggesting that arrhythmias were triggered by air pollution episodes in combination with other factors that increased the

patient's susceptibility to arrhythmia.

Atrial fibrillation is the most common clinically important arrhythmia in older persons and imposes both a large societal burden and economic burden on the health-care system because of decreased quality of life, functional status, and hospitalizations for rhythm management, heart failure, and stroke (Rich et al., 2006).

While an increase in premature supraventricular beats is associated with long-term exposure to fine particle pollution (O'Neal et al., 2017a) and an increase in premature ventricular beats is associated with both short- and long-term exposure to increased concentrations of particle pollution (O'Neal et al., 2017b), the relationship between atrial fibrillation and exposure to particle pollution is less well established. Yet, a recent meta-analysis (Shao et al., 2016) showed an association between short-term exposure to fine particle pollution and the development of atrial fibrillation. The meta-analysis included some individuals with advanced heart disease managed with internal cardiac defibrillators (Link et al., 2013), and the positive association was not limited to fine particle pollution. Atrial fibrillation was also associated with increases in CO, NO and SO2.

Heart Failure: Several epidemiological studies indicate that acute exposures to fine particles contribute to hospitalization and mortality attributed to heart failure (Shah et al., 2013). For example, one large multi-city study conducted in 204 U.S. urban counties examined the association between daily changes in fine particle pollution concentrations and cardiovascular-related hospital admissions. The study reported that the largest association for hospital admissions is due to heart failure (Dominici et al., 2006). The authors reported a 1.28 percent increase in heart failure hospital admissions for a 10 μg/m3 increase in 24-hour average fine particle concentrations.

Stroke: Some studies have reported evidence of an increase in hospitalizations for stroke due to increases in the concentration of ambient fine particles (Wellenius et al., 2012). Recent meta-analyses have provided additional evidence supporting a relationship between both acute and chronic exposures to fine particles and various types of stroke (Shin et al., 2014; Shah et al., 2015). The mechanism for the increase in strokes is not known, but one study found a relatively small but independent effect of higher air temperature, dry air, upper respiratory tract infections, grass pollen, SO2, and suspended particles (Low et al., 2006).

Plaque stability and thrombus formation: Modulation of plaque stability and thrombus formation associated with fine particle exposure is suggested by epidemiological data indicating that the risk of unstable angina and myocardial

infarction may increase by as much as 4.5 percent for each 10 μg/m3 increase in 24-hour average fine particle concentrations (Pope et al., 2006).

What are the chronic exposure effects?

There is accumulating evidence that risk from chronic exposure (months to years) to inhaled fine particles accelerates atherosclerosis and reduces life expectancy.

Atherosclerosis: Several epidemiology studies, including the Multi-Ethnic Study of Atherosclerosis Air Pollution Study (MESA-Air), show that chronic air pollution exposure promotes atherosclerosis. This is indicated by the positive association between chronic particle exposure and an increase in coronary artery calcium (Kaufman et al., 2016), the severity of coronary artery disease (McGuinn et al., 2016), and increased thickness of the internal carotid artery (Künzli et al., 2005; Adar et al., 2013). Animal studies (Suwa et al., 2002, Araujo et al., 2008) have provided insights into the possible mechanisms that include inhibition of the anti-inflammatory capacity of plasma high-density lipoprotein, as well as increases in systemic oxidative stress, systemic inflammation, total amount of lipids in aortic lesions, and plaque turnover and extracellular lipid pools in coronary artery and aortic lesions.

Cardiovascular disease mortality: Fine particle pollution exposure is a risk factor for cardiovascular disease mortality via mechanisms that likely include pulmonary and systemic inflammation, accelerated atherosclerosis, and altered cardiac autonomic function (Dockery et al., 1993). The mechanisms of death associated with exposure to acute and chronic particle pollution are not fully known; however, prothrombotic effects precipitating myocardial infarction and stroke, autonomic instability precipitating arrhythmia, and increased oxidative stress worsening heart failure are speculated to account for the increased risk. Chronic exposure to particle pollution is most strongly associated with mortality attributable to ischemic heart disease, arrhythmia, heart failure and cardiac arrest (Pope et al., 2004).

Several seminal large cohort studies support the association of chronic exposure to air pollution and mortality. The Harvard Six Cities Study (Dockery et al., 1993) and American Cancer Society's Cancer Prevention II Study (Pope et al., 2002) both show an association between chronic exposure to ambient air pollution, particularly fine particle pollution, and an increased risk of death.

The Harvard Six Cities Study found statistically significant associations between chronic exposure to air pollution and mortality (Figure 7), specifically for fine particles and other pollutants strongly correlated with fine particles. Air pollution was also positively associated with cardiopulmonary disease deaths. A follow-up

study (Laden et al., 2006) assessing risk of death after considerable improvement in air quality in these six cities showed that the risk of mortality diminished in proportion to the reduction in air pollution.

The American Cancer Society's Cancer Prevention II Study also assessed the relationship between chronic exposure to fine particle pollution and mortality, but on a national scale (Pope et al., 2002). Similar to the Harvard Six Cities study, the ACS study reported evidence of a positive association between both all-cause and cardiopulmonary mortality and chronic exposure to fine particles.

In contrast to previous studies focusing on mortality in the entire population, Miller and colleagues examined the association between chronic exposure to fine particle pollution and clinical cardiovascular events in post-menopausal women without previous cardiovascular disease (Miller et al., 2007). In this study, one or more fatal or nonfatal cardiovascular event(s) occurred which included death from coronary heart disease or cerebrovascular disease, coronary revascularization, myocardial infarction, and stroke. The authors observed a marked increase in the risk of both cardiovascular events (24% increase), cerebrovascular events (35% increase), and cardiovascular-related mortality (76% increase) in this cohort of women for each 10 μg/m3 increase in the annual average concentration of fine particles.

[www.epa.gov/pmcourse/particle-pollution-and-cardiovascular-effects]

According to Huttunen, et al., short-term exposure to ambient air pollution is associated with increased cardiovascular mortality and morbidity and that this adverse health effect is thought to be mediated by inflammatory processes. They followed elderly individuals with ischemic heart disease. Average ambient PM2.5 concentration was 8.7 micrograms/m3. Of the studied pollutants, PM2.5 was most strongly associated with increased levels of inflammatory markers, most notably with C-reactive protein and IL-12 within a few days of exposure. There was also some evidence of an effect of particulate air pollution on fibrinogen and myeloperoxidase. The concentration of IL-12 was considerably (227%) higher during, rather than before, a forest fire episode. The authors state these findings show that even low levels of particulate air pollution from urban sources are associated with acute systemic inflammation and that particles from wildfires may exhibit pro-inflammatory effects (Huttunen2012).

Dennekamp, et al., found an association between exposure to forest fire smoke and in increase in the rate of out-of-hospital cardiac arrests (Dennekamp2015).

Zhao, et al., did a systematic review and meta-analysis of the impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest. PM10, PM2.5, NO2 and ozone were found to be significantly associated with increase in

out-of-hospital cardiac arrest risk, with the strongest association being observed for PM2.5 (Zhao2017).

Haikerwal, et al., investigated the role of PM2.5 in triggering acute coronary events during the 2006-2007 wildfires in Victoria, Australia. They found PM2.5 exposure was associated with increased risk of out-of-hospital cardiac arrest and ischemic heart disease (Haikerwal(b)2015).

Jones, et al., studied cardiac arrests during California wildfires in 2015-2017 and found that out-of-hospital cardiac arrests increased with wildfire smoke exposure (Jones2020).

Deaths

Johnston, et al., estimated that worldwide exposure to fine-fraction PM2.5 from wildland fires during 1997-2006 were associated with approximately 340,000 deaths per year (Johnston2012).

Faustini, et al., analyzed the effects of wildfires and PM10 on mortality in 10 southern European cities. They found smoke was associated with increased cardiovascular mortality in urban residents, and PM10 on smoky days had a larger effect on cardiovascular and respiratory mortality than on other days (Faustini 2015).

Effects on Children and Pregnant Women

The American Pregnancy Association lists the following as being potential dangers of being exposed to air pollution during pregnancy: Low birth weight, preterm birth, autism, asthma, and fertility problems. Also noted is that particulate matter can cross the placenta and reach an unborn child. (https://americanpregnancy.org/pregnancy-health/how-air-pollution-impacts-pregnancy/)

According to an American Academy of Pediatrics Policy Statement, "*Ambient (outdoor) air pollution is now recognized as an important problem, both nationally and worldwide. Our scientific understanding of the spectrum of health effects of air pollution has increased, and numerous studies are finding important health effects from air pollution at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm birth, infant mortality, deficits in lung growth, and possibly, development of asthma*" (AAPeds2004).

Sraim, et al., reviewed studies looking at possible adverse effects of ambient air pollution on birth outcomes and concluded "*The evidence is sufficient to infer a causal relationship between particulate air pollution and respiratory deaths in the postneonatal period* [1 mo. – 1 yr. of age]". The authors further note that fetuses, in particular, are considered to be highly susceptible to a variety of toxicants, especially during critical windows (sensitive periods of development), because of higher rates of cell proliferation or changing metabolic capabilities (Sraim2005).

Tan-Soo, et al. found that prenatal exposure to smoke from the 1997 Indonesian forest fires resulted in decreased height at age 17. The authors state, "*Because adult height is associated with income, this implies a loss of 4% of average monthly wages for approximately one million Indonesian workers born during this period*" (Tan-Soo2019).

Kunzli, et al., investigated the health effects of the 2003 southern California wildfires on children. The authors found that fire smoke had a substantial effect on children's health. "*All symptoms (nose, eyes, and throat irritations; cough; bronchitis; cold; wheezing; asthma attacks), medication usage, and physician visits were associated with individually reported smoke exposure*." They also note that "*wildfire smoke contains numerous primary and secondary pollutants, including particles, polycyclic aromatic hydrocarbons, carbon monoxide, aldehydes, organic acids, organic compounds, gases, free radicals, and inorganic materials with diverse toxicologic properties" (*Kunzli2006).

According to Vicedo-Cabrera, et al., "*Exposure to wildfire smoke was associated with increased respiratory symptoms in this child population, particularly affecting susceptible individuals with asthma or rhinitis*." (Vicedo-Cabrera2016).

Lim, et al., did a systematic review and meta-analysis of the short-term effect of fine particulate matter on children's hospital admissions and emergency department visits for asthma. They found that children's hospital admissions and emergency department visits for asthma were positively associated with a short-term 10 microgram/m3 increase in PM2.5 (Lim2016).

Cancer

In an October 17, 2013 press release, the International Agency for Research on Cancer (IARC), an agency of the World Health Organization, announced that it had classified outdoor air pollution as *carcinogenic to humans* (Group 1). Particulate matter, a major component of outdoor air pollution, was evaluated separately and was also classified as *carcinogenic to humans* (Group 1)(IARC2013).

Kim and colleagues evaluated the mutagenicity and lung toxicity of particulate matter (PM) from flaming vs. smoldering phases of five biomass fuels (northern red oak, pocosin peat, ponderosa pine needles, lodgepole pine, and eucalyptus). They

found the greatest mutagenicity was for pine. Further, they concluded that smoldering emissions from wildland fires are highly mutagenic and support the notion that smoldering wood smoke is genotoxic and ultimately carcinogenic in humans (Kim2018).

Covid-19

Short-term and long-term exposure to PM2.5 is associated with an increased risk of Covid-19 cases and deaths.

According to the U.S. Centers for Disease Control and Prevention, "*wildfire smoke can irritate your lungs, cause inflammation, affect your immune system, and make you more prone to lung infections, including SARS-CoV-2, the virus that causes COVID-19*." [www.cdc.gov/disasters/covid-19/wildfire_smoke_covid-19.html]

Exposure to particulate matter increases the expression of angiotension-converting enzyme 2 (ACE2) in the lungs which facilitates SARS-CoV-2 viral adhesion.

Wu, et al., found that long-term exposure to air pollution was positively associated with higher mortality rates. They found that for every 1 microgram/m3 increase in PM2.5 exposure, there was an 11% increase in Covid-19 deaths (Wu2020).

Zhou, et al., investigated the number of Covid-19 cases and deaths in California, Oregon, and Washington during the 2020 wildfires. They concluded the overall number of Covid-19 cases and deaths attributable to daily increases in PM2.5 from wildfires was 19,742 and 748, respectively (Zhou2021).

In addition, the CDC also notes that *"[p]eople who currently have or who are recovering from COVID-19 may be at increased risk of health effects from exposure to wildfire smoke due to compromised heart and/or lung function related to COVID-19*." [www.cdc.gov/disasters/covid-19/wildfire_smoke_covid-19.html]

National Ambient Air Quality Standards (NAAQS)

The National Ambient Air Quality Standards (NAAQS) and EPA's associated Air Quality Index (AQI) do not adequately protect public health.

Several studies have found adverse health impacts from exposure to particulate levels below current standards, i.e. at levels AQI considers "*healthy*." There appears to be no threshold level of PM2.5 below which no adverse health effects occur. This has led some researchers to call for revising NAAQS standards.

According to a 2003 report by a World Health Organization (WHO) Working Group:

"Epidemiological studies on large populations have been unable to identify a threshold concentration below which ambient PM has no effect on health. It is likely that within any large human population, there is such a wide range in susceptibility that some subjects are at risk even at the lowest end of the concentration range."

Harvard researchers investigated the association between short-term exposures to ambient fine particulate matter (PM2.5) and ozone, and mortality. They found that in the U.S. Medicare population from 2000 to 2012, short-term exposures to PM2.5 and warm-season ozone were significantly associated with an increased risk of mortality. This risk occurred at levels below current national air quality standards, suggesting that these standards may need to be reevaluated. They also found no evidence of a threshold in the exposure-response relationship below which no increased mortality occurred (Di2017).

Schwartz, et al., investigated the concentration-response relation between PM2.5 and daily deaths. The authors state that several recent articles have reported that exposure to PM10 is associated with daily deaths with no evidence of a threshold. In this study, the authors found an association between exposure to PM2.5 and daily deaths with no level of a threshold down to the lowest levels of PM2.5. They state "*In fact, the curve is quite linear over the exposure range from 0 to 35 micrograms/m3*" and this is consistent with previous results (Schwartz2002).

Fire Accelerant Chemicals

A variety of chemical accelerants are used to start prescribed fires. These chemicals and their breakdown products get into the air and leave residues on the ground. Diesel fuel and gasoline are commonly used to start fires on the ground. Aerial release of ping pong-like balls containing potassium permanganate (KMnO4), ethylene glycol, and polystyrene shells are also used to start fires.

According to the International Chemical Safety Card for potassium permanganate, this chemical "*gives off irritating or toxic fumes (or gases) in a fire*." "*This substance is corrosive to the eyes, skin and respiratory tract*," and " … *may have effects on the lungs. This may result in bronchitis and pneumonia. Animal tests show that this substance possibly causes toxicity to human reproduction or development*" (IPSCpotassiumpermangate).

According to a Risk Assessment of Residues of Fire Accelerant Chemicals prepared for the Intermountain Region USDA Forest Service, Table 1-1, Chemicals Evaluated in Risk Assessment, the residues expected from the use of the above accelerants are diesel fuel, gasoline, MTBE, manganese dioxide, potassium hydroxide, and polystyrene. Styrene is also expected to be released as a gas.

This risk assessment evaluates the risk to humans of drinking contaminated water or fish, and ingesting contaminated soil. It gives recommendations for the quantity

of each kind of accelerant that can be used to avoid harm to humans. It did not, however, assess the human health risk of breathing fire accelerant chemicals (RiskAssessmentResiduesFireAccelerants2002; and companion literature search, LitSearchResiduesFireAccelerants2002).

Although this risk assessment contains useful information, it cannot be relied on to assess the risk to the public of exposure to fire accelerant chemicals because it is out-of-date and does not assess the impact of inhalation of fire accelerant chemicals, the most likely route of public exposure.

Prescribed Fires

According to the U.S. Environmental Protection Agency (EPA), " … *using prescribed fire is not without risk as it can result in smoke related air quality and public health impacts*". In its 2021 report "Comparative Assessment of the Impacts of Prescribed Fire Versus Wildfire (CAIF): A Case Study in the Western United States," the EPA states the goal of the report is to help risk managers take public health impacts of smoke into account when making decisions about using prescribed fire. [www.epa.gov/newsreleases/epa-releases-report-comparing-air-quality-and-public-health-impacts-prescribed-fire]

Even though health impacts from individual prescribed fires (or naturally-occurring fires to which accelerant is added) tend to be lower than those associated with severe wildfires, their cumulative impacts are often similar to or exceed the impact of wildfires, since they occur with much greater frequency.

In Australia, Arriagada et al., examined health impacts from elevated particulate air pollution from 2002-2017. They found that of the total estimated health costs resulting from particulate air pollution, 51% was attributable to prescribed burns and 41% to wildfires (Arriagada2020).

In Georgia, researchers found that the health burden of smoke from prescribed burning is comparable to that estimated for other major emission sectors, such as vehicles and industrial combustion. They say these findings call for greater attention to the air quality impacts of prescribed burning (Afrin2021).

In many ways prescribed fires are similar to wildfires, except they tend to be lower intensity burns that emit greater amounts of particulate matter per unit of biomass burned than wildfires.

"*Unlike wildfires that are of high intensity, prescribed fires are cool low-intensity burns and produce relatively short plumes … While low-intensity prescribed burns (low heat, light emissions) cause minimal risk to life and property, they can however emit large amounts of smoke particulates." "Smoke from prescribed burning can have a substantial impact on air quality and the environment. Prescribed burning is a*

significant source of fine particulate matter (PM2.5 aerodynamic diameter<2.5 micrometers) and these particulates are found to be consistently elevated during smoke events. Due to their fine nature PM2.5 are particularly harmful to human health" (Haikerwal(a)2015).

"*… There is a need to understand the influence of prescribed burning smoke exposure on human health. This is important especially since adverse health impacts have been observed during wildfire events when PM2.5 concentrations were similar to those observed during prescribed burning events* (Haikerwal2015).

According to Ward & Hardy, "*The smoldering combustion phase produces high emissions of particulate matter and CO [carbon monoxide]. Fires of low intensity (those in which the flaming combustion phase is barely sustained) produce high emissions of particulate matter." "For many fuel types, emissions from the smoldering phase overwhelm emissions produced through flaming combustion processes – typical of measurements of smoke from wildfires and during the later stages of prescribed fires"* (Ward&Hardy1991).

Kim et al. found that flaming combustion conditions were more efficient, converting much of the carbon to CO2, whereas more carbonaceous PM and CO (carbon monoxide) were emitted during smoldering. They also found that smoldering pine and pine needles had the highest levels of mutagenicity potencies (Kim2018).

Alves, et al., analyzed smoke from a wildfire in a mixed evergreen forest in Portugal and found that particulate matter and organic carbon emissions were significantly enhanced under smoldering fire conditions (Alves2011).

Navarro, et al, found that PM2.5 concentrations from wildfire smoke were significantly lower than PM2.5 concentrations from prescribed fire smoke (Navaffo2018).

Prescribed fires (and naturally-occurring fires to which is accelerant is added) also differ from wildfires in the application of fire accelerants. These are toxic chemicals that get into the air and can contaminate soil and water. And while prescribed fires can be timed to reduce smoke impacts, the increasing practice of adding accelerant to naturally-occurring fires removes this benefit.

<u>Mitigation</u>

In Air Quality Impacts from Prescribed Forest Fires under Different Management Practices, the authors state that large amounts of air pollutants are emitted during prescribed forest fires. Such emissions and corresponding air quality impacts can be modulated by different forest management practices. These include, but are not limited to, 1) making more use of mechanical thinning to reduce the amount of burning, 2) choosing to burn during seasons that emit fewer pollutants (in Georgia,

equivalent fires in the spring and winter were found to emit more PM2.5 than those in the summer), and 3) better controlling emissions from smoldering by, for example, burning before precipitation (Tian, 2007).

Ravi, et al., investigated the impacts of smoke from prescribed fires on air quality, health, and visibility in protected natural environments. They concluded that a 70% reduction in fire activities would result in significant improvement in air quality in areas in western Oregon, northern Idaho and western Montana where most prescribed fires occur. Using BenMAP, a health impact assessment tool, they showed that several hundred additional deaths, several thousand upper and lower respiratory symptom cases, several hundred bronchitis cases, and more than 35,000 work day losses can be attributed to prescribed fires and these health impacts decrease by 25-30% when a 30% fire emission scenario is considered. The authors also note that as prescribed burning activities become more frequent, they can be more detrimental for air quality and health (Ravi2018).

Environmental Justice

As noted above, people with lower socioeconomic status (SES) are at higher risk of suffering adverse health impacts from air pollution. This can occur because their exposures are higher than those with higher SES. But in addition, for any level of air pollution, they suffer disproportionately more harm. Forastiere, et al., investigated whether social class is an effect modifier of exposure to PM10 (particulate matter with a diameter < 10 microns) and found that their results confirmed previous suggestions of a stronger effect of particulate air pollution among people in low social class (Forastiere2007).

Liu, et al., found increased risks of respiratory admissions from wildfire smoke was significantly higher for blacks than whites (21.7% vs. 6.9%) and stated that their study raised important environmental justice issues (Liu(b)2017).

Executive Order 12898, issued in 1994, established the responsibility of each Federal agency to "*make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low income populations ....*" An accompanying Presidential Memorandum directed that human health, economic, and social effects, including effects on minority communities and low-income communities, be included in the analysis of environmental effects pursuant to NEPA. [https://ceq.doe.gov/nepa-practice/justice.html]

Therefore, analysis of the human health effects of smoke from prescribed fires must also include a breakdown of the severity of those impacts according to socioeconomic status (SES).

Costs

Jones, et al. found "*On average, wildfire smoke in the Western U.S. creates $165 million in annual morbidity and mortality health costs*" (Jones2017).

The costs to the public of exposure to smoke from wildland fires, including prescribed fires, can be considerable. Costs can include medical costs (doctor visits, ED visits, and hospitalization), increases in medication, evacuation costs (transportation, lodging, driver/attendant), purchase of air filters and masks, and lost days of work.

Rappold and colleagues evaluated the health impacts and economic value of wildland fire episodes in the U.S. from 2008-2012. Their models suggest that areas including northern California, Oregon and Idaho in the West, and Florida, Louisiana and Georgia in the East were most affected by wildland fire events in the form of additional premature deaths and respiratory hospital admissions. They estimated the economic value of these cases due to short term exposures as being between $11 and $20 billion (2010$) per year, with a net present value of $63 billion for the 5 years studied (95% confidence intervals $6-$170); and estimated the value of long-term exposures as being between $76 and $130 billion (2010$) per year, with a net present value of $450 billion for the 5 years studied (95% confidence intervals $42-$1,200)" (Rappold2014).

Borgschulte, et al., examined the importance of air pollution from wildfire smoke in the determination of national, annual labor income in the United States. Wildfires account for about 20% of the fine particulate matter emitted in the U.S. They note that air pollution exposure increases infant and elderly mortality and reduces long-run health and future income among those exposed in utero and infancy. Air pollution also negatively affects the broader adult population, for example, by reducing labor supply and productivity.

In summary, this paper found that smoke exposure reduces earnings in both the year of exposure (each day of wildfire smoke exposure caused a roughly linear reduction in labor income of 0.07% in the year of exposure) and the following year, lowers labor force participation, and increases Social Security claiming and payments. With an average of 17.7 days of annual smoke exposure per person, earnings losses sum to 1.26% of annual labor income. They further estimated that the welfare cost of these los earnings is higher than the mortality cost of wildfire smoke (Borgschulte2019).

Kochi, et al., summarized previous studies of the economic analysis of wildfire-smoke-induced health damage, noting that the omission of mortality costs may have resulted in substantial underestimates of total health costs. They further note, "*Work days lost, restricted-activity days, and minor restricted-activity days contribute substantially to total morbidity-related costs, and account for 36 to 74% of total estimated health costs in the studies that did not consider premature mortality.*"

The authors concluded, "*The economic costs of adverse health effects associated with exposure to wildfire smoke should be given serious consideration in determining the optimal wildfire management policy*." "*For example, concerns about adverse health effects from 2008 wildfires in northern California prompted the USDA Forest Service to actively suppress all wildfires in California*" (Kochi2010).